

Vascular complications in high-performance athletes

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Purpose: The purpose of this study was to evaluate our experience with the diagnosis and management of vascular injuries in a group of high-performance athletes.

Methods: Between June 1994 and June 2000, we treated 26 patients who sustained vascular complications as a result of athletic competition. Clinical presentation, type of athletic competition, location of injury, type of therapy, and degree of rehabilitation were analyzed retrospectively.

Results: The mean age of the patients was 23.8 years (range, 17-40). Twenty-one (81%) patients were men, and five (19%) were women. Athletes included 8 major-league baseball players, 7 football players, 2 world-class cyclists, 2 rock climbers, 2 wind surfers, 1 swimmer, 1 kayaker, 1 weight lifter, 1 marksman, and 1 volleyball player. There were 14 (54%) arterial and 12 (46%) venous complications. Arterial injuries included 7 (50%) axillary/subclavian artery or branch artery aneurysms with secondary embolization, 6 (43%) popliteal artery injuries, and 1 (7%) case of intimal hyperplasia and stenosis involving the external iliac artery. Subclavian vein thrombosis (SVT) accounted for all venous complications. Five of the seven patients with axillary/subclavian branch artery aneurysms required lytic therapy for distal emboli, and six required operative intervention. All popliteal artery injuries were treated by femoropopliteal bypass graft with autogenous saphenous vein. The external iliac artery lesion, which occurred in a cyclist, was repaired with limited resection and vein patch angioplasty. All 12 patients with SVT were treated initially with lytic therapy and anticoagulation. Eight patients required thoracic outlet decompression and venolysis of the subclavian vein. Thirteen arterial reconstructions have remained patent at an average follow-up of 31.9 months (range, 2-74). One patient with a popliteal artery injury required reoperation at 2 months for occlusion of his bypass graft. Eleven of the patients with an arterial injury were able to return to their prior level of competition. All of the patients with SVT have remained stable without further venous thrombosis and have returned to their usual level of activity.

Conclusions: Athletes are susceptible to a variety of vascular injuries that may not be easily recognized. A high level of suspicion, a thorough workup including noninvasive studies and arteriography/venography, and prompt treatment are important for a successful outcome. (J Vasc Surg 2001;33:935-42.)

High-performance athletes are susceptible to a variety of vascular injuries. Athletes who are exposed to repetitive motion or high-speed collisions may have either arterial or venous injuries.¹⁻³ Extremity pain in the active athlete seldom invites a diagnosis of vascular compromise at the time of initial evaluation. The sports medicine literature is replete with descriptions of musculoskeletal abnormalities and injuries in the active athlete, but rarely are arterial or venous abnormalities addressed. Other institutions have presented their experience with vascular injuries in this group of patients, and it is apparent that this is an increasing problem as athletes become more highly trained.¹⁻⁶ Failure to recognize a vascular injury in these patients may result in the failure to treat a potentially limb-threatening lesion. Proper diagnosis requires a high level of suspicion, a detailed vascular history and physical examination of the

patient, and a thorough workup including noninvasive studies and arteriography/venography when indicated.

This study was designed to review our experience with a large group of young, competitive athletes with either an arterial or a venous injury resulting from athletic competition. We evaluated the patient's clinical presentation, athletic competition, type of injury (venous or arterial), treatment, and degree of rehabilitation.

PATIENTS AND METHODS

From June 1994 to June 2000, we treated 26 patients who sustained vascular complications as a result of athletic competition. Patients were enrolled prospectively in a vascular registry and their clinical courses retrospectively reviewed. Patients were divided into two groups according to whether they had a venous or arterial injury. All venous lesions consisted of subclavian vein thrombosis (SVT). Arterial injuries were grouped according to the location of the lesion: axillary/subclavian artery and popliteal artery or iliac artery.

Patients with SVT. The clinical diagnosis of primary venous thrombosis (Paget-Schroetter syndrome) was based on each patient's medical history and the results of a physical examination and a venous duplex study. When SVT was demonstrated with duplex ultrasound scan, the patient was admitted to the hospital, and a contrast venogram from the level of the proximal forearm to the innominate-superior vena caval confluence was per-

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formed. Attention was given to the region of the shoulder girdle, and images were obtained with the arm in the neutral position and in abduction and external rotation.

Following a venogram with positive findings, catheter-directed thrombolysis was initiated. Initially, urokinase (Abbokinase; Abbott Laboratories, Abbott Park, Ill) was used as the primary lytic agent for thrombolysis. Since 1999, alteplase (a recombinant tissue plasminogen activator; Activase; Genentech, South San Francisco, Calif) has been used. Infusions lasted from 24 to 72 hours depending on the success of thrombolysis as demonstrated with serial venograms. Heparin was infused simultaneously through the sidearm of the infusion sheath at rates from 500 to 1200 U/h to obtain an activated partial thromboplastin time of 60 to 80 seconds. Thrombolysis was stopped when one of the following criteria was met: no interval change in the appearance of the vein in two sequential venograms, any bleeding complication, biochemical evidence of systemic thrombolysis or disseminated intravascular coagulopathy, or 72 hours of continuous infusion.

At the conclusion of thrombolysis, the patency of the vein was assessed with a completion venogram. Any positional impingement of the subclavian/axillary vein or collateral veins was noted. The patients' medication was converted from intravenous heparin to warfarin, and they were discharged when the international normalized ratio reached therapeutic levels. Patients were instructed to engage in normal activities of daily living, but to avoid athletic competition while taking warfarin sodium (Coumadin).

Patients were reevaluated at 4 weeks with color flow venous duplex scanning. They were interviewed about any symptoms they had while they rested or exercised and were examined for evidence of venous hypertension. Indications for surgical intervention included persistent symptoms of venous hypertension and significant obstruction of the subclavian vein or collaterals with abduction and external rotation or recurrent venous thrombosis. Thoracic outlet decompression was accomplished through a single supraclavicular incision. The details of our approach have been described previously. The first rib was resected in all cases, and fibrous bands impinging on the subclavian vein were released. Care was taken not to divide any major collateral veins. A complete circumferential venolysis of the subclavian vein completed the procedure.

Postoperatively, patients underwent anticoagulation for 1 month. Physical therapy was started on the second postoperative day, and patients were allowed to gradually resume full physical activity at 6 weeks. Balloon angioplasty was reserved for symptomatic residual stenosis after surgical decompression, but was not required in this particular group. Patients were followed up on a biannual basis that included a venous duplex scan.

Axillary/subclavian artery. Patients with axillary/subclavian artery injuries presented with relatively subtle symptoms, which were frequently unrecognized as vascular problems at the time of their initial evaluation. Typical symptoms included a decrease in speed or

control during pitching, coolness of fingers, or loss of endurance during athletic activity.

Five of seven patients were treated with 24 hours of catheter-directed thrombolysis to dissolve clot in the digital vessels. This was followed by operative exploration of the aneurysmal vessel and appropriate repair. Two patients were treated with surgery alone. Repair consisted of resection of the aneurysm and either lateral repair of the artery with a vein patch or vein interposition graft. Patients were allowed to resume normal activities postoperatively, but were instructed to avoid strenuous activity for 6 weeks. At that time they started an aggressive physical therapy regimen specific to their athletic activity and were advanced to their normal activity over a 2- to 3-month period.

Popliteal artery. Each patient's medical history and results from a physical examination as well as noninvasive Doppler studies were used to diagnose popliteal artery injuries. If the diagnosis of popliteal artery injury was suspected, an arteriogram was performed. Three of six patients had posterior knee dislocations and presented with acute ischemia. The remaining three patients presented with chronic ischemia. All patients were treated with a femoral to below-knee popliteal bypass graft with a reversed saphenous vein graft. Patients were discharged home, physical therapy was administered, and they were allowed to resume athletic activity primarily on the basis of how they recovered from the orthopedic component of their injury. All patients were evaluated with graft surveillance at 1 month and then on a biannual basis.

Iliac artery. One patient had a lesion of the external iliac artery that resulted from competitive cycling. The patient was an Olympic-class cyclist with increasing times in his trials resulting from fatigue in his left thigh at high speeds. Results of a physical examination and ankle/brachial indices (ABIs) at rest were normal. Noninvasive testing immediately after exercising to the point of producing symptoms, however, revealed that his left leg ABI decreased to 0.39. An arteriogram demonstrated an area of stenosis and redundancy in the external iliac artery with otherwise normal arteries.

The patient was explored through a left lower quadrant retroperitoneal incision. The external iliac artery was completely mobilized from small branches that were tethering the artery to the psoas muscle. The elongated artery was shortened and the stenosis repaired with a saphenous vein patch angioplasty. He was instructed to avoid any strenuous activity for 6 weeks. He was followed up at 1 month with repeat noninvasive testing before and after exercise and then biannually.

RESULTS

Between June 1994 and July 2000, we treated 26 patients who sustained vascular complications attributable to their athletic competition. The patients' mean age was 23.8 years (range, 17-40). There were 21 men (81%) and five (19%) women. Athletes included 8 major-league baseball players, 7 football players, 2 world-class cyclists, 2 rock climbers, 2 wind surfers, 1 swimmer, 1 kayaker, 1 weight

Table I. Summary of results

	No. (%)	Age (y) (range)	Sex (M/F)	Lytic therapy	Surgery (%)	No symptoms at last follow-up	Patent	Return to previous level of competition
Venous	12/26 (46%)							
SVT	12 (100%)	23.3 (16-25)	9/3	12/12	8/12 (67%)	12/12 minimal	12/12	12/12
Arterial	14/26 (54%)							
Axillary/subclavian	7 (50%)	25.7 (19-32)	7/0	7/7	6/7 (86%)	7/7	7/7	7/7
Popliteal	6 (43%)	22.3 (17-40)	5/1	0	6/6 (100%)	6/6	6/6, primary 5/6	3/6
Iliac	1 (7%)	26	1/0	0	1/1 (100%)	1/1	1/1	1/1

Table II. Athletic competition and type of injury

Patient no.	Diagnosis	Operation	Sport	Follow-up (mo)
1	SVT	None	Swimming	30
2	SVT	Rib resection, venolysis	Wind surfer	26
3	SVT	Rib resection, venolysis	Baseball	25
4	SVT	None	Football	24
5	SVT	Rib resection, venolysis	Football	23
6	SVT	Rib resection, venolysis	Marksman	15
7	SVT	None	Rock climber	15
8	SVT	Rib resection, venolysis	Rock climber	6
9	SVT	None	Football	6
10	SVT	Rib resection, venolysis	Baseball	6
11	SVT	Rib resection, venolysis	Baseball	6
12	SVT	Rib resection, venolysis	Football	4
13	Axillary/subclavian artery	Resect aneurysm, lateral repair	Baseball	74
14	Axillary/subclavian artery	Resect aneurysm, lateral repair	Baseball	74
15	Axillary/subclavian artery	Resect aneurysm, lateral repair	Baseball	52
16	Axillary/subclavian artery	None	Baseball	47
17	Axillary/subclavian artery	Resect aneurysm, vein interposition	Cyclist	46
18	Axillary/subclavian artery	Resect aneurysm, lateral repair	Volleyball	5
19	Axillary/subclavian artery	Resect aneurysm, vein interposition	Baseball	2
20	Popliteal artery	Fem-pop	Football	71
21	Popliteal artery	Fem-pop	Football	58
22	Popliteal artery	Fem-pop	Kayaker	50
23	Popliteal artery	Fem-pop	Weight lifter	35
24	Popliteal artery	Fem-pop	Wind surfer	6
25	Popliteal artery	Fem-pop 3/9/00, fem-pop 6/27/00	Football	2
26	Iliac artery	Vein patch angioplasty	Cyclist	7

Fem-pop, Femoropopliteal; *SVT*, subclavian vein thrombosis.

lifter, 1 marksman, and 1 volleyball player. There were 14 (54%) arterial and 12 (46%) venous complications. Arterial injuries included 7 (50%) axillary or subclavian artery or branch artery aneurysms with secondary embolization, 6 (43%) popliteal artery injuries, and 1 (7%) case of intimal hyperplasia and stenosis involving the external iliac artery. SVT accounted for all venous complications.

Patients with SVT. Twelve (46%) of 26 patients had SVT as a result of athletic competition (Table I). The mean age of this group was 23.3 years (range, 16-25). There were nine male and three female patients. The distribution of their athletic activities is outlined in Table II. None of these patients had a cervical rib. All 12 patients received thrombolytic therapy after the diagnosis of SVT. Eight of the 12 patients had complete venographic evi-

dence of clot lysis, and four patients had partial (> 50%) lysis. The patients with only partial lysis were all patients who underwent lytic therapy at least 4 weeks after their thrombotic event. After lytic therapy, patients were treated with 4 weeks of outpatient anticoagulation. Eight (67%) of the 12 patients required thoracic outlet decompression and subclavian vein venolysis according to the operative criteria described above. There was no significant morbidity or mortality in the surgical group. The average postoperative length of stay was 2.1 days (range, 2-3). Venous duplex imaging at 4 weeks demonstrated continued patency of the subclavian vein in all patients in the surgical group, and all patients had resolution of their symptoms. The average length of follow-up in this group of patients was 13.9 months (range, 4-26).

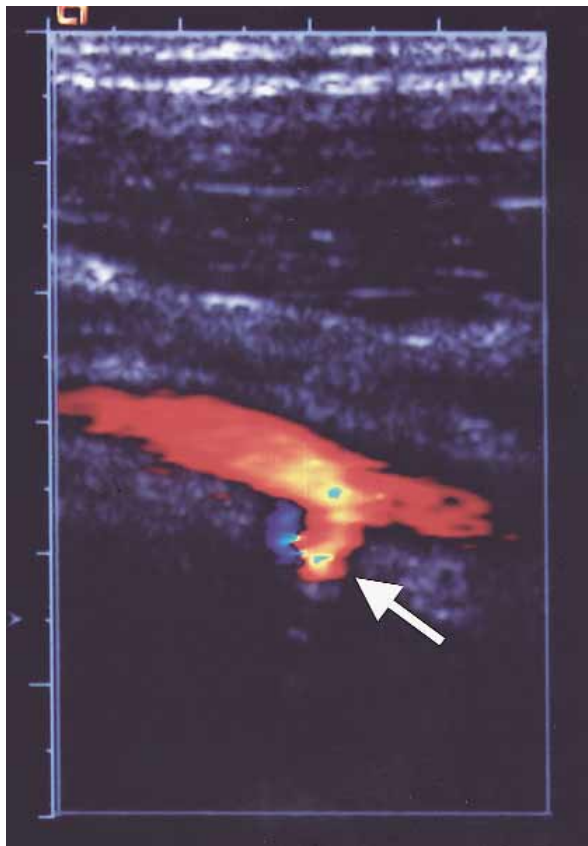


Fig 1. Noninvasive imaging of axillary artery that shows presence of aneurysm in branch artery. This study prompted an arteriogram to evaluate for distal embolization.

Four patients were treated conservatively. These patients underwent anticoagulation for an average of 3.6 months (range, 2-7). All had minimal symptoms at the conclusion of their anticoagulation. To date, these patients' subclavian veins have remained patent, as revealed with duplex ultrasound scan, and patients have been able to return to their prior level of competition. The length of follow-up in this group was 18.8 months (range, 6-30).

Patients with axillary/subclavian artery injuries. Of the 14 patients with arterial injuries seven (50%) had aneurysms of the proximal upper extremity arteries: subclavian artery (1), axillary artery at the level of the circumflex humeral (1), circumflex humeral artery (3), subscapular artery (1), and suprascapular artery (1). The mean age of patients in this group was 25.7 years (range, 19-32) (Table I). All of the patients were men. Five patients were baseball players (4 professional and 1 collegiate). There was one collegiate volleyball player and one cyclist (Table II). Arterial pulses in these patients were normal, although the Allen's test revealed evidence of fixed digital occlusions. Two patients had ischemic digital ulcers. Noninvasive studies demonstrated diminished pho-

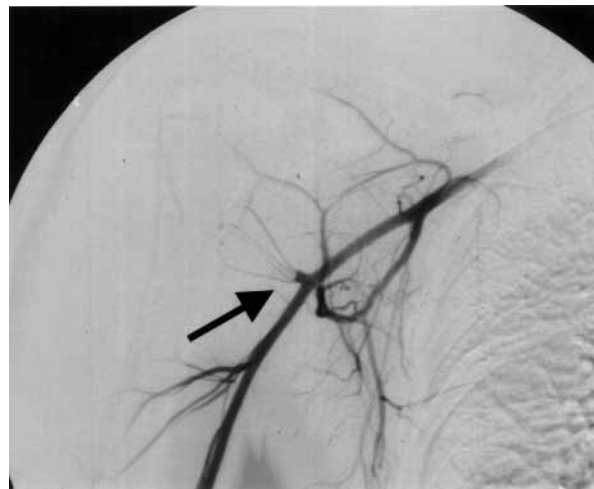


Fig 2. Arteriogram of collegiate volleyball player who complained of coolness in his fingertips after any overhead smashes. Arteriogram demonstrates branch artery aneurysm with evidence of distal embolization.

tolethysmographic waveforms in the digits of the affected extremity. Color duplex imaging demonstrated a branch artery aneurysm in five of these patients (Fig 1). Arteriography was used to demonstrate the site of the aneurysm and the extent of embolization (Fig 2). Five of seven patients had arteriographic evidence of significant distal emboli on their arteriogram. These patients received preoperative lytic therapy before undergoing operative management. Surgical intervention was recommended to all seven patients. Six (86%) of seven patients chose to undergo surgical resection of their aneurysm. One patient chose conservative management with 6 weeks of antiplatelet therapy and arm rest. Of the six patients who underwent surgical therapy, four had resection of the aneurysm and lateral repair of the artery with a vein patch angioplasty. The other two patients had resection of the aneurysm and repair of the artery with a reversed interposition segment of greater saphenous vein.

All arterial reconstructions have remained patent to date with a mean follow-up of 42.2 months (range, 2-74). Photoplethysmographic waveforms reverted to normal in all patients by 6 months. The six surgical patients have been able to return to competition without further symptoms. The seventh patient retired from pitching.

Patients with popliteal artery injuries. Six (43%) of the 14 patients with arterial injuries had popliteal artery injuries (Table I). The mean patient age was 22.3 years (range, 17-40). There were five males and one female. Three patients had posterior knee dislocations and had associated acute popliteal artery injuries that were repaired on an emergency basis. This group included two football players and one kayaker. The other three patients presented with symptoms of unilateral lower extremity weak-

ness and discomfort with physical exertion. This group included 1 football player, 1 weight lifter, and 1 windsurfer. All patients underwent noninvasive studies diagnostic of popliteal artery obstruction. This was confirmed with arteriography that demonstrated short segments of occlusion of the popliteal artery (Fig 3). All patients underwent femoropopliteal bypass grafting with a reversed saphenous vein graft. In two of six patients this was done by means of a posterior approach, and in the other four patients a medial approach was used.

Five of the six grafts have remained patent with a mean follow-up of 44.0 months (range, 6-71). In one football player who had activity-limiting chronic leg fatigue while running, a femoropopliteal bypass graft with a short segment of reversed saphenous vein was performed through a posterior approach. This graft occluded within 3 months presumably for technical reasons and the severe inflammatory reaction that was encountered in his popliteal fossa. The patient subsequently underwent a successful femoropopliteal bypass graft through a medial approach.

Three (50%) of six patients could return to their previous level of activity. The three patients with the posterior knee dislocations were unable to achieve their prior level of competition because of their musculoskeletal injuries.

Patients with iliac artery injuries. One patient had an external iliac artery stenosis (Fig 4). His postoperative course was uncomplicated, and he returned to training 6 weeks after surgery. Postoperatively, his ABIs at rest were normal, and after exercise his ABI on the left side improved to 0.75. The ABI on the right side after exercise was 0.85, and the patient had no complaints of leg fatigue with exercise. The patient has since gone on to make the US Olympic cycling team.

DISCUSSION

A vascular lesion should be suspected in any athlete complaining of fatigue, loss of endurance, pain, or swelling in an extremity. This is especially true in athletes who are involved with repetitive activity. Several reports have documented vascular lesions occurring in these athletes.¹⁻⁹

In our group of patients, 19 of 26 had problems related to the upper extremity. Twelve had SVT, and the other seven patients had problems with aneurysms of the subclavian or axillary arteries or their branches. Of this group of patients there was a direct correlation between repetitive upper extremity use and vascular injury in 16 of the 19 patients (Table II).

SVT resulting from thoracic outlet syndrome is well documented. Reports have focused on numerous etiologies, including supernumerary ribs, compression of the artery and vein between the scalene muscles, and compression between the clavicle and first rib during the act of hyperabduction of the arm.¹⁰⁻¹² Recently, we reported our results with management of subclavian vein thrombosis.¹¹ In that report 41% of patients did not require surgery for the treatment of SVT. Our patients were treated initially with thrombolysis and anticoagulation. Surgical intervention was reserved for those patients with persistent symp-



Fig 3. Arteriogram of 18-year-old high school football player with short-segment occlusion of popliteal artery, who complained of fatigue and pain in his leg with running. This was repaired with femoropopliteal bypass graft with reversed saphenous vein graft.

toms of venous hypertension or documented significant obstruction of the subclavian vein or collaterals with abduction and external rotation of their upper extremity. Athletes with SVT were treated by means of the same algorithm. The four patients with SVT who did not undergo surgery remained symptom free after anticoagulation.

In 1986 Fields et al⁸ documented the significance of upper extremity arterial injuries in athletes with the report



Fig 4. Arteriogram of Olympic-class cyclist demonstrating subtle stenosis of external iliac artery. Notice reduced luminal diameter of external iliac artery as compared with common femoral artery. There is also irregularity to the contrast along luminal surface of external iliac artery.

of a baseball pitcher who had a stroke after thrombosis of his right subclavian artery with subsequent propagation of the clot into the innominate and right carotid arteries. Injury to the subclavian artery is usually a complication of thoracic outlet syndrome and results from compression of the artery by a cervical rib, from an anomalous first rib, or by the soft tissue structures of the thoracic outlet. Repetitive injury to the subclavian artery may produce arterial stenosis that can progress to arterial occlusion, as in Fields et al's patient. Subclavian stenosis may also be associated with poststenotic dilatation of the artery or frank aneurysm formation. These dilated areas may accumulate thrombus that can be a source of distal embolization.^{1,2,4-9}

Injury to the axillary artery may be caused by compression by the pectoralis minor tendon or by compression of the axillary artery by the head of the humerus during hyperabduction and external rotation. Stretch injury to the branches of the axillary artery, the circumflex humeral arteries and the subscapular artery, also may occur with repeated hyperabduction.⁶⁻⁸ Reekers et al⁹ identified posterior circumflex humeral aneurysms in three professional volleyball players. All had evidence of thrombus in the aneurysm and distal emboli. McCarthy et al¹ have described similar findings in 11 athletes with either branch artery aneurysms or subclavian aneurysms. The presumed cause of the aneurysms in these reports and in our seven patients was repetitive trauma to the vessel during exercise. It is postulated that while the shoulder is hyperextended and externally rotated, the humerus compresses

the axillary artery and stretches its branches. It is this stretching of the artery that creates forces that weaken the arterial wall and causes the artery to become aneurysmal. The humeral head subsequently compresses these aneurysms during hyperextension, pushing thrombus into the axillary arterial stream.^{1,2}

Correct diagnosis of this entity requires a high index of suspicion that is based on predisposing physical activity and detailed imaging. On the basis of our experience and previous reports, it seems wise to suggest that individuals who repeatedly perform strenuous shoulder rotations and demonstrate signs and symptoms of upper-limb ischemia undergo noninvasive testing and arteriography, as indicated, to exclude any abnormalities. Surgical intervention leads to resolution of symptoms and allows athletes to return to their previous level of competition.

The vascular anatomy of the lower extremity predisposes the popliteal area to traumatic injury because of the tethering at the adductor hiatus and the soleus arch. Knee dislocations should be followed with arteriography or exploration of the vessels. Epiphyseal separations of the proximal tibia and distal femur and fractures about the knee should be observed closely for 24 to 48 hours.^{3,13,14} The etiology of claudication in the athletic population requires a careful history and physical examination and may require vascular studies to determine if the etiology is one of popliteal artery entrapment, adventitial cystic disease of the popliteal artery, or adductor canal outlet syndrome.

Several reports have described cases of external iliac artery injury in professional cyclists. Most cases have involved cyclists who have ridden more than 150,000 km. The left external iliac artery is affected more often than the right. The pathologic lesion has been described as endofibrosis with marked thickening of the intima. The lesion is thought to be a result of repetitive injury to the vessel.¹⁵⁻¹⁷ It is thought that the enlarged psoas muscle, the flexed position of the cyclist, and the branch vessels tethering the external iliac artery to the psoas combine to produce elongation and stenosis of the artery. The diagnosis cannot be appreciated on the basis of a resting examination. However, ABIs performed after exercise, to the extent that symptoms are produced, will demonstrate a significant decrease, typically to less than 0.5. Most authors have described repairing the lesion by dividing the branches to the psoas and shortening and patching the external iliac artery. Endarterectomy and vein graft replacement have also been reported. Repair with prosthetic graft or angioplasty has not been successful.^{16,17} The outcome of repair of these lesions has been excellent results in published reports and in our own patient.

CONCLUSIONS

Athletes are susceptible to a variety of vascular injuries that may not be easily recognized. A high level of suspicion, a thorough workup including noninvasive studies and arteriography/venography, and prompt injury-specific treatment are important for a successful outcome.

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DISCUSSION

Dr Frank R. Arko. With regard to Dr Dille's first question, the delay in treatment of the thoracic outlet patients: last year Dr Anthony Lee presented the protocol for treatment of our patients. Our protocol basically is patients who received thrombolysis are then anticoagulated for a month and then are seen again at that time. We feel that not all patients with a subclavian vein thrombosis require surgery. In our series 40% did not require surgery.

The reason for the delay is it allows some of the inflammation to disappear prior to operation. It makes the surgery technically easier. Again, we only operate on patients who have persistent symptoms of venous hypertension or who have evidence of obstruction with upper arm extremity maneuvers.

With regard to his next question, when do we use thrombolytics in the patients with upper extremity branch artery aneurysms, early in the series the first couple of patients did not receive thrombolysis, but the last five have all received thrombolysis. Now, these are patients who present with both acute and chronic symptoms of distal emboli.

So it's certainly not going to completely clear up all of the emboli, but all five who underwent thrombolysis were noted to have had some improvement on their post-thrombolytic arteriogram. So we feel it's important but certainly not necessary, and we have not had any problems with it.

As for the role for screening for these patients, that has been looked at in the past by Dr Michael Rohrer in Massachusetts for patients with repetitive upper arm use, basically in pitchers. He found that since it is such a low incidence of this defect that the overall role for screening is probably limited.

Dr G. Eugene Strandness (Seattle, Wash). Several months ago I received a call from my vascular laboratory. They called me and said there was a Canadian doctor there with his wife and they wondered if I would come down and see her. He couldn't get her taken care of in Canada so he came down to Seattle.

Anyway, it turned out she was a world-class cyclist who could no longer cycle anymore because she developed left upper-thigh

claudication. I reviewed a lot of papers for circulation on this injury, and they mainly come from France and they're very confusing. Most of them are rejected, I think, because of the lack of scientific content.

Anyway, this girl was really interesting because at rest the only thing you could find was a little bit of disturbed flow on her external iliac artery. When you put her on a treadmill and really pushed her, the amount of flow disturbance she got was tremendous.

Then I told her that I would be happy to take care of her in Seattle, but he went back to Vancouver and fortunately was able to get a hold of one of our members, David Taylor, who operated on her and put a vein patch on her external iliac artery. Within 3 weeks she qualified for the Olympics. Her name is Carol Montgomery, and in fact, if you ever get a hold of the Canadian version of *Time* magazine, her picture is in there. So it will be very interesting to see whether or not she really does this well in the Olympics.

The other professional athlete I was just indirectly involved with was Oil Can Boyd, who actually thrombosed his subclavian axillary artery by subluxing the head of his humerus every time he pitched and, of course, this ruined his career. He was thrombolized twice. His artery was entirely normal, but that ended his career. Thank you.

Dr Arko. Thank you. Again, with regard to the cyclist, this is not an injury that you would see in just your normal average cyclist. Normally it's been reported that most of these cyclists have cycled well over 150,000 km, and the reason for the pathology is as yet unknown. Some believe it's the position of the cyclist. In their flexed position they get an enlarged soleus muscle, and sometimes you get an enlarged branch that goes to that soleus muscle, which tethers that artery. Others feel it's the increased flow in the artery with increased sheer pressures that causes the injuries.

Dr Kaj Johansen (Seattle, Wash). I remained confused a bit about what we should be doing about effort thrombosis of the subclavian vein. Members of this society have promulgated with excel-

lent data the proposal that all such patients should undergo thoracic outlet decompression. Yet other members recently, and David Taylor's name has been mentioned by Dr Strandness, David Taylor's group in Vancouver as well as Neil Olcott and others have proposed that, in fact, we don't need to be doing this potentially disfiguring and damaging procedure. Your data, in fact, tend to support that in this circumstance.

As recently as last week a major-league pitcher who had undergone thoracic outlet decompression was reported to have rethrombosed his subclavian vein. I do a lot of these operations, and I remain unsure about whether I ought to be or not.

My question here is in those patients who are not operated upon but simply observed. Are they allowed to go back to doing the exertion that brought on this problem, and for how long, especially in patients who might be doing some sort of physical contact during their athletic activity? Are they anticoagulated?

Dr Arko. Those are both very good questions. Of the patients in the group that we looked at, there were four patients who were treated nonoperatively. They were kept on anticoagulation slightly longer than the operative group. Basically, it was 3 months versus just a little over a month in the operative group. They were basically kept with limited activity for 6 weeks. They were allowed to go back to their normal activities but refrained from any physical activity using that upper extremity for 6 weeks.

Now, the patients in the operative group were allowed basically to start physical therapy the day after surgery. So there was some slightly perceived decrease in time to a physical activity in the operative group, but again, with such a small series it's hard to state anything scientifically.

Dr Fred Weaver (Los Angeles, Calif). Regarding the subject of screening for baseball pitchers, we have prospectively screened 18 major-league pitchers in spring training using duplex scanning and found that shoulder instability correlated with a significant reduction in volume flow over the course of a 50-pitch workout. It may be that pitchers with shoulder instability should be targeted for screening of arterial problems by either PPG or duplex scanning.

Dr Arko. Again, I think just a high index of suspicion in patients who present to the sports physician now that this injury is becoming more well-known should just prompt quick intervention.

Dr Mark Nehler (Denver, Colo). A couple of questions. One, were there specific types of pitchers? I mean, was it the type of pitch they were throwing that was rotating their arm in a specific way that was consistent with all the pitchers?

We operate on a lot of elderly folks and divide muscles and things with a band. There are specific techniques when you're operating on high-performance athletes where you try to preserve things that in a normal exposure to say, a fem-pop or something, you would divide tendons that you wouldn't divide normally in those patients.

Dr Arko. With the pitchers, not all of the patients with subclavian artery or axillary artery or branch aneurysms were pitchers. There was actually one volleyball player and a world-class mountain biker. Again, I can't say if there was one particular pitch that caused them to have that, but it was basically with any type of pitch they threw that would cause coolness in their fingers because the humeral head would press down on the branch artery

aneurysm. With regard to the technical aspects of the operation, we try to preserve all the tendons as much as possible.

Dr Peter Lawrence (Irvine, Calif). Just one last question from me to clarify. In patients who have a major arterial injury in these high-performance athletes that is corrected, what is the advice that's given and what is it based upon as to returning to work? I know that David Cone has been a well-publicized athlete, but there are a number of them. You just reported a cyclist who returned to high performance within a period of time and presumably has the same anatomic situation uncorrected.

If an anatomic defect is not corrected or the cause of the arterial injury is not corrected, how do you make the decision as to whether they should return to high-performance activity?

Dr Arko. Well, normally the patients are kept at rest after any operation for basically 4 to 6 weeks and then allowed to return to a progressive physical regimen.

With regard to the cyclist, he was operated on, and the injury was repaired by shortening the artery and a vein patch angioplasty. We had to go through a retroperitoneal approach through the left flank, and so from that aspect we requested that he not partake in any aggressive physical activity for 4 to 6 weeks.

With these aggressive athletes, it's sometimes hard to do that, and he stayed in shape basically with electrostimulation to his legs and abdominal muscles while he was at rest. After those 4 to 6 weeks, he gradually got back onto his therapy.

Dr Lawrence. I understand that you're correcting the arterial injury. My question has to do with the underlying cause of the arterial injury. If you do not correct that, do you expect a high recurrence rate, or why are you advising these people to go back to high performance? Don't you anticipate that they're going to have a similar problem in the future?

Dr Arko. Actually, there have been some series again in France on these world-class cyclists who have been operated on with very good results, and actually, the risk of recurrence in their series has been fairly low at 3%. It can occur, but he's willing to take that risk to ride in the Olympics.

Dr Cornelius Olcott. I agree with what Gene said with the patient that he had, and the French have also commented on this. You do free up that iliac artery off the soleus. They think that the branch arteries that are present in these cases, tethering that to the soleus, have to be divided to free that up so that it doesn't keep getting traumatized as much.

I share some of Gene Strandness' concern about the French literature, and I am skeptical of it, but I think that they have shown, at least I've been convinced on the basis of a couple of these cases now, that this is a real injury.

As far as the upper extremity athletes go, once you get rid of that aneurysm, like Ralph presented also, then you don't have that toothpaste effect where when they do pitch, there's compression of that aneurysm, which allows embolization. So I think you have to change something to prevent these folks from getting problems again.

Does that answer your question?

Dr Lawrence. That does answer my question, yes. I mean, Oil Can Boyd, obviously he's repeatedly doing the same thing and rethrombosing. Nothing has been corrected that caused the original problem, and you would anticipate that there would be a high recurrence rate if you don't correct some anatomic abnormality that is causing the problem.